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By

PATRICIA JEAN GAMBERA
B.S. (Montclair State College) 1986

THESIS

Submitted in partial satisfaction of the requirement for the degree of

MASTER OF SCIENCE
in

Nutrition

in the

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ABSTRACT

USE OF THE FOOD GUIDE PYRAMID TO IMPROVE DIETARY INTAKE AND REDUCE CARDIOVASCULAR RISK IN ACTIVE DUTY AIR FORCE MEMBERS. P.J. Gambera, RD, P.A. Davis, PhD, and B.O. Schneeman, PhD, Departments of Nutrition and Clinical Nutrition, University of California, Davis, CA

The goal of this study was to determine if adherence to the Food Guide Pyramid guidelines, when combined with exercise, results in significant reductions in cardiovascular risk and enhancement in aerobic capacity (VO_2 max) when compared to a regimen of exercise therapy alone. Twenty men and twelve women, mean age=32, with similar baseline VO_{2max}, total cholesterol (TC), low density lipoprotein (LDL), body mass index (BMI), and dietary fat intakes were randomized into two groups. All subjects participated in a 90-day fitness improvement program. One-half of the subjects (treatment group), received weekly, individualized dietary counseling using the Food Guide Pyramid as an educational tool. All participants completed a food frequency questionnaire at baseline and post-treatment. Those in the treatment group maintained three-day food records weekly throughout the study to monitor compliance and assess changes in food intake. Substantial changes in diet occurred for the treatment group from baseline. Mean percentage of fat calories decreased from 39% to 23% while mean servings from each of the food groups changed to reflect current guidelines: Milk (1.6 to 2.6), Meat (4.5 to 3.0), Vegetable (2.0 to 2.6), Fruit (0.8-3.6) and Bread (5.3 to 7.5) servings, respectively. The treatment group also experienced significant reductions in TC, LDL, and BMI, of 9% (p=0.003), 13% (p=.005) and 2% (p=0.0001), respectively. Significant improvements in fitness, as estimate by VO_{2max} (p=0.01) of 14% in the control and 38% in the treatment group were achieved. We conclude that dietary modification in accordance with the Food Guide Pyramid and the Dietary Guidelines for Americans results in significant reductions in known cardiovascular risk factors and improves the response to exercise training.

LITERATURE REVIEW

INTRODUCTION:

Coronary heart disease (CHD) affects approximately 7 million Americans and causes about 1.5 million heart attacks and 500,000 deaths a year (1). Estimates of the 1991 morbidity data from CHD and heart attack cost the country $44.5 billion in lost productivity and health care services (2).

There is strong and consistent evidence for the relationship between saturated fat intake, high blood cholesterol and increased risk for CHD (3). Clinical, animal, and epidemiological studies demonstrate that high intakes of saturated fatty acids (SFA) increase the concentration of serum total (TC) and low-density lipoprotein cholesterol (LDL-C). CHD morbidity and mortality is positively related to LDL-C (4) and remains the primary target of cholesterol-lowering therapy (5). Lowering LDL-C in asymptomatic individuals with hypercholesterolemia reduces CHD deaths and nonfatal coronary events (6, 7).

Saturated fat intake is the major dietary determinant of serum TC and LDL-C concentration in populations. In the United States, 35 to 37% of calories are derived from fat sources; there are virtually no differences with age and gender (8). Saturated fat consumption averages 12 to 15% of total calories, and for boys and girls ages 2 to 11, this value exceeds 14% (8).

About 26% of the adult population is above a body weight for health, and the prevalence of overweight has not declined in this country for two decades. Overweight is associated with elevated serum cholesterol levels, elevated blood pressure, and non insulin-dependent diabetes and is an independent risk factor for CHD (3, 9).

Epidemiological studies suggest that consistent exercise could have significant individual and public health benefit for CHD prevention (10). Unfortunately, few Americans engage in regular physical activity despite the potential benefits (11).

Healthy People 2000 is a document of measurable targets with a goal to improve the health of the Nation by the end of the decade (11). In view of their role in risk reduction, nutrition and exercise objectives were included in this health promotion strategy. Thus, the primary focus of this literature review will be on the relationship between dietary quality, particulary fat, and its impact on the regulation of lipoproteins and CHD risk. In addition, the role of exercise and fitness improvement will be explored as it relates to lipoprotein metabolism and CHD prevention. The format for this review includes four major sections: ***Section I*** introduces dietary lipid terminology, followed by fat digestion and absorption in humans. ***Section II*** discusses lipoproteins and their role in lipid transport and cholesterol homeostasis. ***Section III*** explores the impact of diet on lipoprotein concentrations. ***Section IV*** reviews exercise training, cardiorespiratory fitness and lipoprotein response.

LIPID TERMINOLOGY:

Lipids are compounds that are not soluble in water but are soluble in non polar solvents such as ether and benzene. The bulk of any lipid molecule is non polar. Structurally, the lipids are quite diverse; there is no common subunit in their structure. The primary building blocks in human lipids are fatty acids, glycerol, shingosine and sterols (12, 13).

Fatty acids all have a single carboxyl group at the end of a hydrocarbon chain. The most natural fatty acids have an even number of carbon atoms. The hydrocarbon chains can be either saturated (lacking carbon-carbon double bonds) or unsaturated (12, 13).

Triglycerides (TG) are the most prevalent group of lipids and considered a neutral fat. Three fatty acids esterified to one glycerol backbone make up a TG moiety. Generally, fatty acids are ingested in the form of TG but they are hydrolyzed in the course of digestion and the free fatty acids utilized for TG re-synthesis (12, 13).

Saturated fatty acids (SFA) contain fatty acids lacking carbon-carbon double bonds. SFA can be synthesized by the liver and other tissues from protein, carbohydrate or fat. Fats containing a high portion of SFA are usually solid at room temperature. They may be of natural origin or synthetic. In general, such fats are of animal origin, and thus contain cholesterol. However, some vegetable oils such as the "tropical" oils of coconut, palm and palm kernel contain very high

percentages of SFA. The two most abundant SFA for human consumption are palmitic acid (C16) and stearic acid (C18) (12, 13).

Hydrogenated fats are vegetable fats containing fatty acids that have had their unsaturated double bonds saturated by catalytic hydrogenation using platinum or nickel. Hydrogenation produces some fatty acid isomers with an abnormal trans configuration as opposed to the normal cis configuration. It has been suggested that these trans fatty acids are more atherogenic than their cis isomers (14).

Monounsaturated fatty acids (MFA) contain fatty acids with one carbon-carbon double bond, usually between carbons 9 and 10. Oleic acid (C18) and palmitoleic acid (C16) comprise the bulk of the MFA for human consumption. Under normal circumstances, oleic acid is not converted into other fatty acids, nor is it a starting material for prostaglandin production. It occurs in generous amounts in all lipid containing foods whether animal or vegetable. Animal fats vary from 20-40% oleic acid and vegetable oils from 12% for safflower oil to 75% for olive oil (12, 13).

Polyunsaturated fatty acids (PUFA) contain fatty acids with two or more double bonds. They are liquid at room temperature and are generally of vegetable origin. PUFA include linoleic acid (C18) with two double bonds, linolenic acid (C18) with three, and arachidonic acid (C20) with four double bonds. Linoleic acid (omega-6 family) and linolenic acid (omega-3 family) are termed essential fatty acids because they cannot be synthesized by

mammals. Arachidonic acid is produced in humans from linolenic acid (12, 13).

Cholesterol is the major sterol in the body and precursor to all the steroid hormones. The source of cholesterol is both the diet and liver. The liver can use protein, carbohydrate or fat to synthesize cholesterol. The human liver makes roughly 0.5 g of cholesterol per day. Synthesis is regulated by exogenous sources. When dietary cholesterol increases, the liver compensates by producing less (12, 13). A more complete summary of cholesterol regulation is located elsewhere in this review.

LIPID DIGESTION AND ABSORPTION:

Digestion. The process of lipid digestion begins immediately at ingestion, yet the majority of digestion of TG occurs in the small intestine. After consuming a fat-containing meal, the process of emulsification and enzymatic hydrolysis begins even prior to reaching the stomach. Gastric contraction provides the necessary force to break up food particles and fat globules into smaller droplets. In the stomach, gastric lipase partially hydrolyzes TG into free fatty acids, glycerol, and mono- and diglycerols. Phospholipases remove fatty acids from phosphoglycerides. Bile salts solubilize or emulsify this mixture of free fatty acids, monoacylglycerols, and diacyglycerols into droplets less than 1 micron in diameter termed micelles. These micelles transport lipids through the bulk aqueous phase of small intestinal contents (12, 13).

Absorption. Humans in general absorb fat very efficiently. The upper intestine of the average person absorbs over 90% of the lipid ingested. A pH of 6-7 is required to optimize enzyme action and fat absorption. Once the lipid micelles are brought into contact with the mucosal cell membrane of the proximal jejunum, the cholesterol, monoglycerides, fatty acids and vitamins diffuse into the lipid cell membranes. The residual, largely bile salt micelle, recycles. Inside the intestinal mucosal cells, TG are re-synthesized and combined with B-lipoproteins, phosphoglycerides, and cholesterol to form chylomicrons. The chylomicrons then

enter the lymphatics and travel through the thoracic duct to reach the blood stream (12, 13).

Unlike the long-chain fatty acids, the medium- and short-chain fatty acids (<12 carbons) are well absorbed without bile salts. They enter the portal venous system directly and travel to the liver, instead of traveling through the lymphatics as TG in chylomicrons. Bile salts also emulsify cholesterol to facilitate its absorption. Once absorbed, cholesterol is esterified to unsaturated fatty acids to create cholesterol esters (12).

LIPID TRANSPORT:

Lipoproteins are water-soluble complexes of high molecular weight that are composed of lipids (cholesterol, TG, phospholipids) and one or more specific proteins called apolipoproteins (apo). All lipoproteins share a similar micelle structure in which the hydrophilic moieties of the phoshpolipids, cholesterol, and apolipoproteins are arranged at the surface and the hydrophobic TG and cholesterol esters are oriented toward the interior to form the core (15). Each class of lipoprotein has a specific function determined by its point of synthesis, lipid composition, and apolipoprotein content. There are five major classes of lipoproteins: chylomicrons, very low-density lipoproteins (VLDL), intermediate density lipoproteins (IDL), low-density lipoproteins (LDL), and high-density lipoproteins (HDL). HDL are further sub fractionated into HDL_2 and HDL_3 and LDL into small and large particles.

Lipoprotein metabolism is complex and is a function of the relative rates of synthesis, degradation, and exchange among the lipoprotein classes. Conceptually, there are three lipoprotein metabolic systems: exogenous fat transport, endogenous fat transport and reverse cholesterol transport (16, 17).

EXOGENOUS FAT TRANSPORT:

Chylomicrons are the largest of the lipoproteins and the least dense, containing a high proportion of TG with small amounts of cholesterol, phosholipid and protein. Chylomicrons are synthesized in the smooth endoplasmic reticulum of epithelial cells that line the small intestine. The particles enter lacteals in the intestinal villi and are transported through the thoracic duct into the blood. Nascent chylomicrons contain apo B-48, apo A-1 and apo A-IV and acquire apo C and E in the blood from HDL. After a fatty meal, the blood appears milky due to the high concentration of chylomicrons. Apo C-II activates lipoprotein lipase in the capillaries of adipose, heart, skeletal muscle, and lactating mammary tissue, hydrolyzing TG and releasing free fatty acids to these tissues. When most of the TG is depleted, the chylomicron is considered a remnant but still contains cholesterol, apo E, and apo B-48. Some surface material is transferred to HDL, and the remaining remnant is transported through the bloodstream to the liver where it is taken up and recycled (12, 13).

ENDOGENOUS FAT TRANSPORT:

Very Low Density Lipoproteins are TG-rich lipoproteins, synthesized in the liver, and packaged with specific apolipoproteins. In addition to their large proportion of TG, VLDL contains some cholesterol and cholesterol esters, apo B-100, apo C-I, apo C-II, apo C-III, and apo E. These lipoproteins are transported from the liver to the adipose tissue where activation of lipoprotein lipase by apo C-II causes the release of free fatty acid for the adipocyte. Here, TGs are re-synthesized and stored as intracellular lipid droplets. Through this process VLDL is transformed to (IDL) (12, 13).

Intermediate Density Lipoproteins are thought to be a metabolic product of VLDL and the precursor to LDL particles. Apo B-100 is conserved during the transformation of VLDL to IDL. Excess surface material from IDL is transferred to HDL. Two possible fates exist for IDL. It can be taken up by the liver directly through the LDL receptor, or it can be converted to low density lipoproteins (LDL) (12, 13).

Low Density Lipoproteins are further converted from IDL through the loss of TG. In contrast to the TG-rich chylomicron and VLDL, LDL contains mainly cholesterol and cholesterol esters. Apo B-100 remains, conserved from the original VLDL particle (12). LDL functions to carry cholesterol to peripheral tissues that have specific surface receptors that recognize apo B-100 (18). These receptors mediate the uptake of cholesterol and cholesterol esters. Chemical modification of the arginine contained in apo B

by means of cyclohexane increases the half-life of the circulating LDL (19, 20). This observation indicates the significance of apo B in the catabolism of LDL.

REVERSE CHOLESTEROL TRANSPORT:

High Density Lipoproteins are small protein-rich particles, synthesized in the liver and intestine, with the important function of cholesterol transport. High protein and low TG contents make them the most dense lipoprotein. HDL contains several apolipoproteins including apo A-I, apo A-II, apo C-I, apo C-II, apo C-III and apo E, as well as the enzyme lecithin-cholesterol acyl transferase (LCAT). Newly synthesized (nascent) HDL collects cholesterol esters from other circulating lipoproteins through the action of cholesterol ester transfer protein (CETP). This protein facilitates the transfer of cholesterol ester from HDL to VLDL, LDL, and to a lesser extent, chylomicrons in exchange for TG. Chylomicrons and VLDL are both rich in cholesterol and phosphatidylcholine after the action of lipoprotein lipase and removal of their TG. LCAT, on the surface of HDL, converts the phophatidylcholine and cholesterol to cholesterol esters. The esters then enter the interior of the nascent HDL and changes its shape from a flat disc to a mature HDL sphere. This cholesterol-rich, mature lipoprotein returns to the liver where the cholesterol is unloaded and may be converted into bile salts (12, 13).

CHOLESTEROL REGULATION:

Approximately 65-70% of TC is transported in the LDL. Thus, the catabolism of LDL is an important component in the homeostatic regulation of endogenous cholesterol. Brown and Goldstein received the Nobel Prize for their proposed mechanism of cholesterol homeostasis involving LDL receptors (18).

LDL Receptor Mediated Endocytosis. Each LDL particle circulating in the bloodstream contains one apo B-100 which is recognized by target tissue LDL receptors. The binding of LDL to a receptor initiates endocytosis, bringing the LDL and associated receptor into the cell. The LDL components undergo lysosomal hydrolysis, resulting in the intracellular release of cholesterol. These receptors perform the key function of regulating cholesterol metabolism in a number of tissues and preventing an over accumulation of cholesterol in the cell. Approximately 70% of LDL is removed from plasma via this mechanism. The remaining LDL is modified in plasma and removed by scavenger receptors on macrophages and endothelial cells (13).

Plasma LDL concentration is influenced by the cells' need for cholesterol. In mammals, cholesterol concentration is largely regulated by intracellular cholesterol concentration. High intracellular cholesterol causes reduced production of the LDL receptors, slowing the uptake of LDL which can lead to high concentration of LDL in the plasma (18). When the sum of the cholesterol synthesized in the liver and peripheral tissues and that obtained in the diet exceeds the amount required by bodily

tissues, pathologic accumulations of cholesterol in the blood can occur leading to an accelerated rate of atherogenesis (13).

Plasma TC, LDL-C and HDL-C are strong predictors of CHD risk (21). Plasma TC and LDL-C are positively related to CHD risk and HDL-C negatively related to CHD risk. HDL_2 is a better predictor of CHD risk than either total HDL-C or HDL_3 (22). Smaller LDL are thought to be more atherogenic than larger LDL particles (23). There is increasing evidence that serum apolipoproteins, particularly the ratio of apo A-1 to apo B may be the most consistent discriminator of the severity of coronary artery disease (24).

Presently, the National Cholesterol Education Program Adult Treatment Panel (NCEP) recommends assessing serum TC and HDL-C in initial risk assessment when accurate testing is available (5). If TC is higher than 6.2 mmol/L (240 mg/dL), a lipoprotein profile is indicated. A LDL-C level of 3.4-4.1 mmol/L (130-159 mg/dL) places an individual at risk and a level $\geq$ 4.1 mmol/L (160 mg/dL) indicates high risk for CHD (5). HDL-C levels < 0.9 mmol/L (35 mg/dL) is a risk factor and 1.6 mmol/L (60 mg/dL) or greater is considered protective (5).

DIET AND PLASMA CHOLESTEROL:

The relationship between diet and CHD has received extensive investigation since the famine years of World War I and II. Fewer heart attacks were observed among the starving European populations during those years. When dietary fat was diverted to ammunition production in Norway during World War II, death from coronary artery disease decreased (25). Since the end of World War II the relationship between diet and plasma lipid levels has been consistently demonstrated throughout the world (26-38).

Saturated fatty acids. The hypercholesterolemic effects of SFA has been elucidated through years of sound scientific research (3, 26). The clear connection between SFA and CHD has led to the current recommendations that SFA should contribute less than 10% of total calories (5) and less than 7% for the treatment of elevated plasma TC and LDL-C (29).

All SFA may not produce equal adverse effects on blood lipids. Fats high in lauric acid, myristic acid, or palmitic acid (12, 14 and 16 carbon atoms, respectively) have been shown to increase the cholesterol concentration (30), whereas fatty acids with 4 to 10 carbon atoms have no effect. Stearic and oleic acids (18 carbons) have been found to effectively lower blood cholesterol when either replaces palmitic acid in the diet (31).

Early studies on the effects of trans fatty acids on blood lipids produced conflicting results. Some studies (32-34) suggested that partially hydrogenated trans fatty acids, as

compared to their natural cis isomer (e.g. oleic acid), elevated serum TC concentration, while others (35-37) could not confirm this. Recent studies suggest that trans fatty acids are hypercholesterolemic compared with their cis isomer (14, 38). Although the rise in LDL-C is less than that associated with C12-16 saturated acids, a diet high in trans fatty acids may contribute to a reduction in HDL-C (38).

Polyunsaturated fatty acids. Two predictive equations, developed by Keys et al. (39) and Hegsted et al. (40), determined that the dietary SFA raises cholesterol twice as much as the cholesterol lowering effect of omega-6 fatty acids. Linoleic acid is the major PUFA in the diet. It appears that PUFA may independently reduce blood cholesterol concentration through the increased expression on hepatic LDL receptors (41). Evidence suggests that high doses of linoleic acid can induce a decrease in LDL-C as well as an unfavorable lowering of HDL-C (41-47) and apo A-I (48).

Omega-3 polyunsaturated fatty acids, primarily provided by fish and fish oils, may provide a protective effect against CHD. Epidemiological studies in the 1970s (49), comparing the diets of the Greenland Eskimos and Caucasian Danes found mortality from myocardial infarction for the Eskimos to be one-tenth that of the Danes despite similar total intakes of fat. A closer comparison of the two diets revealed substantial differences in the quality of fat consumed. The Eskimos' diet was high in omega-3 fatty acids while the Danes consumed twice as much saturated fat.

When taken in sufficient quantities, omega-3 fatty acids appear to reduce VLDL-TG synthesis (50). However, LDL-C concentration may rise as plasma TG falls (51, 52), in individuals with known hyperlipidemia. When the omega-3 fatty acid replaces SFA in the diet, LDL-C drops (53), but this effect is not seen when SFA remains constant (54). Additionally, substitution of omega-6 with omega-3 fatty acids in normal lipemic individuals can cause a drop in HDL-C. However, a rise in HDL-C occurs when used in the treatment of hypertriglyceridemia (52). One of the most beneficial effects of omega-3 fatty acids on CHD may be related to their anti-thrombotic properties (55). In general, the evidence suggests that omega-3 fatty acids are protective, particularly when substituted for SFA in the diet. Based on its association with a decreased incidence of CHD, it seems prudent to recommend the inclusion of fish that are rich in omega-3 fatty acids One investigator reported beneficial effects from just one or two fish meals per week (45).

Monounsaturated fatty acids. In Keys' classic Seven Countries Study (26), it was noted that people living in southern Italy and Greece had a low incidence of CHD despite their consumption of relatively high fat diets. A closer examination of the fat quality revealed that the diet was high in olive oil, which is rich in oleic acid (a primary dietary MFA), and low in SFA. MFA have since been shown to lower plasma TC and LDL-C concentrations when substituted for SFA in the diet (43).

Oleic acid is the most common MFA in the human diet and is considered neutral in its influence on TC because it has an effect

similar to that of carbohydrates (56, 57). When compared with palmitic acid, oleic acid was found to lower plasma concentrations of total and LDL-C without causing a decrease in the concentration of HDL-C (42, 43, 56, 57). Mensink and Katan had different findings in their study of subjects who consumed a diet containing varying amounts of MFA (15.1% vs. 10.8%), PUFA (7.9% vs 12.7%) and similar SFA (12.9% vs 12.6%). In men, HDL-C fell slightly in both groups; in women, HDL-C was unchanged (44). The effects of MFA and PUFA on HDL-C remains inconclusive (41, 44, 47) and warrants further investigation.

Dietary cholesterol. One of the earliest correlations between dietary cholesterol and plasma cholesterol levels was reported by Connor et al. in a study of Tarahumara Indians (58). Controlled metabolic ward studies (39, 40, 59) and later cross-sectional population studies (60, 61) have confirmed a direct relationship between dietary cholesterol and blood cholesterol levels. The plasma incremental response to dietary cholesterol is greater at intakes less than 500 mg/d (62). It has been estimated that by decreasing dietary cholesterol from 750 mg to 250 mg/d, an individual consuming 2500 kcal would lower the blood cholesterol level by 0.21 to 0.39 mmol/L (63, 64).

At least three factors have been identified in the relationship between dietary cholesterol and lipoprotein response: (1) composition or quality of the dietary fat, (2) baseline dietary cholesterol intake, and (3) individual variability (65). An experiment designed to examine the variability in the plasma response to egg feeding found a large range (-0.44 to

+1.38 mmol/L) of plasma cholesterol in response to 28 days of adding three eggs daily to a habitual diet (66). In two similar experiments conducted with free-living subjects, the heterogeneity of the plasma lipid response to manipulations of fat quality and cholesterol quantity was again confirmed (67, 68).

Even though there is variability in the plasma cholesterol response to dietary cholesterol, most individuals responds to predictive mathematical models (63). In a 2,500 kcal diet, a 100 mg increase in dietary cholesterol increases plasma total-C about 4 mg/dl (63).

Dietary fiber. Evidence suggests that specific foods rich in fiber may have beneficial effects on several CHD risk factors, including hyperlipidemia, high blood pressure, obesity, and diabetes (69, 70). The National Cancer Institute recommends that the public increase dietary fiber levels to 20-30 g/d with an upper limit of 35 g/d (71). Presently, the average American dietary fiber consumption from food is 8 g for women and 10 g for men (72).

A number of epidemiological and clinical studies have shown that sources of soluble fiber, such as oats, legumes, pectin, psyllium, and selected gums, have a beneficial effect on plasma lipid levels. Results of a meta-analysis (73) indicated that oat products supplying 3 g of soluble fiber per day or more were effective in reducing TC. In a double-blind study of low-dose consumption, the addition of 34 g of oat bran daily in healthy medical students reduced TC and LDL-C significantly while HDL-C remained constant (74). In a large study using healthy

volunteers, a 5% reduction in TC was achieved after consuming a fat modified diet for 6 weeks. When either 39 g of oat bran per day or 35 g of oatmeal per day was added to the low-fat diet in place of carbohydrate, a further 3% decrease in TC was found after 6 weeks (75). Tinker et al. found the addition 6 g of dietary fiber from a fruit, prunes, to the usual diet of free-living hypercholesterolemic men caused significant lowering in LDL-C (76). Additionally, Haskell et al. observed significant reductions in TC and LDL-C in healthy subjects supplemented with a combination of psyllium, pectin, guar gum, and locust bean gum only when the total fiber supplement was 15 g/d. (77). In a recent literature review on soluble fiber and serum lipids, Glore et al. found TC was significantly reduced in 68 of the 77 (88%) studies reviewed (78). The authors conclude that soluble fiber has the potential to lower serum TC and LDL-C levels.

Proposed mechanisms attributed to the hypolipidemic action of fiber include the following: (1) binding of bile acids, causing a demand for cholesterol to replenish bile acid pools, thus reducing serum cholesterol concentrations; (2) fermentation of soluble fiber by colonic bacteria resulting in a production of short-chain fatty acids that influence lipid metabolism (79, 80); (3) increased catabolism of LDL-C (81, 82); and (4) indirect effects as consumption of high fiber foods reduces dietary energy, saturated fat and cholesterol (83, 84).

SUMMARY:

The relationship between diet and CHD is strong (26-28). The clearest connection is linked to the hypercholesterolemic effect of SFA (3, 5, 26, 29), particularly fats high in lauric, myristic and palmitic acid (30). Some studies have suggested that trans fatty acids elevate serum TC (32-34) while other studies have not confirmed this relationship (35-37). PUFA may independently reduce blood cholesterol levels by increasing hepatic LDL receptors (41). However, high doses have been shown to lower HDL-C (41-44). Evidence supports the protective qualities of omega-3 fatty acids (50, 52, 55), particularly when substituted for SFA (53). In contrast to PUFA, MFA are shown to lower plasma TC and LDL-C (43) while preserving the concentration of HDL-C (42, 43, 56, 57). Dietary cholesterol increases plasma TC (39, 40, 59-61), but the response is variable (65-68). Evidence suggests that certain types of soluble fiber may facilitate the reduction in lipoprotein concentration (74-78), although the mechanism of action remains elusive (79-84).

PHYSICAL ACTIVITY AND HEALTH:

Physical activity is defined as any bodily movement produced by skeletal muscles that results in caloric expenditure (85). A growing body of evidence suggests that regular physical activity is associated with a reduced risk of CHD (86-89). Physically inactive people are almost twice as likely to develop CHD as people who engage in regular physical activity (90). An association also exists between physical activity and physical fitness levels as measured by maximal oxygen uptake and heart rate response to submaximal exercise (91, 92). A higher level of fitness seems to correlate with a more favorable risk profile for CHD (21, 93, 94), and appears to be a major contributor to the delay in all-cause mortality (95). Independent of conventional coronary risk factors, lower levels of physical fitness are associated with higher risk of death from CHD and cardiovascular disease (CVD) in clinically healthy men (96). Epidemiological studies suggest that a weekly expenditure of 1000 calories could have significant individual and public health benefit for CHD prevention especially for those who are originally sedentary (10). Despite the known benefits, less than 10 percent of the U.S. adult population exercise at a level recommended by the 1990 physical fitness and exercise objectives: "exercise which involves large muscle groups in dynamic movement for periods of 20 minutes or longer, 3 or more days per week, and which is performed at an intensity of 60 percent or greater of an individual's cardio-respiratory capacity (97)."

CARDIORESPIRATORY FITNESS:

Cardiorespiratory fitness or aerobic capacity, describes the bodies ability to perform moderate intensity activity for a prolonged period of time without undue stress or fatigue (98). Higher levels of cardiorespiratory fitness can be achieved by increasing the frequency, duration, or intensity of the activity, but the relationship is not linear (98). Studies reveal that vigorous physical activity that is required to achieve and maintain cardiorespiratory fitness can also contribute substantially to energy expenditure, and probably provide additional protection against CHD over less vigorous forms or regular physical activity (88, 95).

VO2max. An important index of fitness and exercise tolerance is the amount of oxygen that can be taken up by working muscles during maximal exercise (99). Ideally, a graded exercise test that includes measurement of ventilation and gas exchange to determine maximal oxygen uptake (VO_{2max}) is used to measure aerobic function. However, this method is technically difficult, time consuming and costly. Consequently, tests have been developed which estimate VO_{2max} from physiologic responses to measured submaximal exercise. Several investigators have developed equations which predict VO_{2max} from treadmill test duration (100-103) based on the rationale that VO_2 is linearly related to test duration.

The most popular methods of submaximal testing have been those which predict VO_{2max} by extrapolation from the relationship

of submaximal VO_2 and heart rate values to an assumed age-related maximal heart rate (104, 105). Cycle ergometry is one method of testing which is easy to administer, provides accurately quantifiable work rates (106) and yields submaximal VO_2 values that are less variable than those obtained from treadmill exercise (107). There is an independent relationship of cycle ergometry estimates of VO_{2max} to age, gender and/or body size that can cause ergometry to underestimate VO_{2max} by up to 10 percent (105, 108). Correction factors may be used to adjust the predicted VO_{2max} value for these variables (104, 105, 107, 109); however, the influence of age and stature is most evident within the 5th percentiles of height and age (110). Although some have shown 10-27% error with high standard of error estimates using submaximal tests (111, 112, 113), Storer et al. have generated equations to predict VO_{2max} to within 10% of its true value in 95 out of 100 subjects (105).

Specificity of Training and Testing. Questions regarding specificity of training mode were address in a study by Rathnow et al. The authors concluded that training in a conditioning program can become sufficiently variable that expected increases in aerobic power are not produced (114).

During submaximal exercise testing, the specificity of treadmill (TM) testing in trained runners is greater than the specificity of cycle ergometry (CE) estimates of VO_{2max} in the same group of runners. Cycle VO_{2max} scores will not improve as much as TM estimates if running is the training mode. However, persons who use a cycle for training improve VO_{2max} on both TM

and cycle tests. The difference between TM and cycle estimates for VO_{2max} is less if training is accomplished on a cycle (115).

EXERCISE AND LIPOPROTEINS:

A large body of evidence has suggested that exercise has a dramatic effect upon lipid metabolism, specifically TG and HDL-C. TG uptake from circulation is highly related to muscle LPL activity. Training may increase the capacity to clear TG from circulation by increasng LPL activity in plasma and in parenchymal cells in muscle (116). Postparandial lipemia is also found to be less in trained versus untrained subjects (117). The link between TG and CHD appears to be complex, and it may be explained by the association between high TG, low HDL-C, and unusually atherogenic forms of LDL (5). Epidemiological studies have shown a strong independent and inverse relationship between HDL-C and CHD (118-120). The most important lifestyle factors reported to affect HDL-C are alcohol, smoking, diet composition, body weight, and physical activity (121).

Several investigators have reported increases in HDL-C with exercise training (122-125). Wood et al. reported a dose-response relationship between the amount of exercise performed and plasma HDL-C (126). Others have reported no change (127-130), or a decrease (131, 132) in HDL-C when controlling for diet quality and weight loss.

Exercise effects on LDL-C have generally been small, both in cross-sectional comparisons of athletes and sedentary persons, and in longitudinal training studies. Similarly LDL-C concentrations do not change appreciably with acute exercise in the absence of weight change or dietary modification (133).

However, Williams et al. observed considerably lower concentrations of small LDL particles when runners were compared to sedentary controls (23).

Although the direct effect of exercise on lipoproteins appears to be limited to HDL, adiposity is closely related to habitual exercise, lipoprotein pattern, and lipase activities (134). The Framingham investigators have noted a correlation between obesity and lipoproteins. LDL-C levels were directly correlated, while HDL-C inversely correlated with body weight (134). Training programs almost always result in the loss of body fat, and a reduction in body fat may lead to a more favorable lipoprotein profile. In one study, a reduction in body weight due to exercise training led to reductions in plasma TC and LDL-C of 0.34 and 0.29 mmol/L, respectively (135).

SUMMARY:

A growing body of evidence suggests that regular physical activity is associated with a reduced risk of CHD (86-89). Physical activity is also related to physical fitness (91, 92), and a higher level of fitness is correlated with an improvement in CHD risk profile (21, 93, 94).

Lipoprotein response to exercise training has largely been limited to TG and HDL-C. Several investigators have reported an increase in HDL-C with exercise (122-125) that appears to be a dose-response relationship (126). When controlling for diet quality and body weight, the increase in HDL-C with exercise may be lost (127-132). Perhaps the greatest indirect effect of exercise on the reduction of plasma lipoproteins is related to weight loss (135).

RATIONALE

Increased plasma low density lipoprotein cholesterol (LDL-C) concentration and decreased high density lipoprotein cholesterol (HDL-C) have been correlated with increased risk of CHD (4, 5, 115, 118).

Most severe forms of hypercholesterolemia (LDL-C > 6.7 mmol/L [260 mg/dL]), in the absence of obvious disorders that might produce a secondary elevated cholesterol concentration are the result of genetic disorders of lipoprotein metabolism (5). In the absence of genetic disorders of lipid metablolism, cardioprotective diets that are low in saturated fat have been found to facilitate the reduction of plasma total cholesterol and low density lipoproteins (41, 136, 137). Individuals with genetic forms of hypercholesterolemia may not satisfactorily respond to dietary modification without the addition of drug therapy (5).

Clinical trials have demonstrated that lowering serum cholesterol will reduce new coronary events and mortality in patients without established CHD (5). It has been calculated that the risk of fatal coronary heart disease is 32% lower in individuals consuming a cardioprotective diet than in those following a typical American diet, and life expectancy is 5 years greater in the former group (138). Several investigators have shown that by decreasing the intake of saturated fats and dietary cholesterol and increasing the intake of polyunsaturated fats and food providing soluble fiber, plasma cholesterol levels can be reduced by up to

29% and LDL-C by over 33% (139-141). In most studies, levels of HDL-C are not altered by standard lipid-lowering diets.

However, when the effects of weight loss by dieting or running were studied on HDL subfractions in sedentary, moderately overweight men, dieters had a mean decrease in HDL-2b relative to the exercisers or controls. In other studies, moderate exercise, as part of a hypocaloric, low saturated fat, low cholesterol diet, was found to improve concentration of HDL-C (125, 142). In a recent study, the effect of exercise was found to be additive when combined with a prudent diet in decreasing TC, LDL-C and TG (143).

The National Cholesterol Education Program (NCEP) currently recommends a low saturated fat, low cholesterol diet, with weight loss if indicated, to correct elevated cholesterol concentration (5). The Dietary Guidelines for Americans were revised in 1990 as general guidelines for a healthy diet for those over the age of two (144). Changes were made to provide the public with the best, most current advice for choosing a healthy diet. The Food Guide Pyramid was developed as part of this revision to serve as a daily food guide, with an intent to help individuals put the Dietary Guidelines into action. The message of the Pyramid is to consume at least the minimum number of servings from five major food groups: the milk, yogurt and cheese group; the meat, poultry, fish, dried beans, eggs and nut group; the vegetable group; the fruit group; and the bread, cereal, rice and pasta group. However, it is quite possible to satisfy the suggested number of servings from each of the food categories with a diet that is excessive in calories,

total fat, saturated fat, cholesterol, sodium and simple sugars. A conscious effort to choose leaner, fiber rich, nutrient dense foods within each category is essential to ensure the Dietary Guidelines for Americans are satisfied.

Although the benefit of diet on the reduction of CHD risk is well documented, to date, little research has been conducted using the Food Guide Pyramid as an educational tool. Additionally, insufficient quantitative data are available to accurately measure the potential cardioprotective benefits of adhering to a prudent diet within this framework.

Although there is a clear, independent association between therapeutic diets and exercise on the reduction of lipoproteins and CHD risk, it is not known whether adherence to the Food Guide Pyramid, which promotes the inclusion of fruits, vegetables and whole grains and a switch to leaner varieties within each food category, can have similar cardioprotective effects. It is also unclear if adherence to a healthy eating plan can alter the fitness outcome of an aerobic conditioning program.

To explore these unanswered questions, we selected United States Air Force members as our target group. In 1992, the Air Force Nutritional Medicine and Base Food Services joined efforts to initiate a campaign entitled "Check it Out" to heighten the awareness of healthful food choices throughout the military installation. As part of this campaign, the Food Guide Pyramid became a point of sale to market nutritional information. Food Guide Pyramid literature, posters, and table-tents were disseminated throughout the base dining and medical treatment

facilities. This campaign was partially built on the premise that provision of nutritional information was sufficient to effect change in eating behavior.

In conducting this research, *our goals* were to determine if: 1) dietary modification consistent with the Dietary Guidelines for Americans using the Food Guide Pyramid as an educational tool can result in larger reductions in known CHD risk factors when compared to exercise therapy alone; and 2) if dietary modification can enhance the response to exercise training and fitness outcome.

Our results would indicate whether individualized dietary counseling, using the Food Guide Pyramid as an educational tool, can facilitate changes in diet over time that are consistent with the Dietary Guidelines for Americans. Additionally, we would have a more accurate measure of the potential dietary effects on plasma lipoprotein values, fitness outcomes, and CHD risk prevention.

MATERIALS AND METHODS

OVERVIEW:

Air Force Fitness. Each Air Force member is expected to maintain an acceptable level of fitness for health and well being and to ensure total force readiness in case of war and/or national emergency. The *Air Force Fitness Program* is designed to administer aerobic testing and provide motivation, support and education to maximize individual aerobic carrying capacity. Individuals who do not meet the minimum standard of fitness are entered into a structured, supervised program to help improve their aerobic function.

Aerobic fitness testing. In 1992, the cycle ergometer replaced the mile and half run to measure an individual's fitness level. The cycle ergometer is considered a tool to predict an estimated aerobic fitness level. This method of testing has proven to be a valid and reliable predictor of VO_{2max} (105, 113).

Prior to administering each test, the cycle was calibrated and the seat height adjusted so the leg was fully extended with a slight bend at the knee during the down stroke. Subjects were fitted with a transmitter around the chest to monitor heart rate response. Predicted VO_{2max} was determined by collecting heart rates against increased resistance over a short duration of time during submaximal effort. The work rate was determined by a

computer (*User's Guide for the Air Force Cycle Ergometry Test for Estimating Aerobic Capacity* , version 3.1; April 1993, Human Systems Center, Brooks Air Force Base, TX) based on age, gender, activity level, height, weight and whether one smokes or not.

Individuals then pedalled at a set rate, keeping pace with a metronome set at 50 bpm, against a known resistance for the duration of the test. The computer may direct the work rate to be increased in the first three minutes to bring the subject up to a work rate that is necessary to achieve a steady state heart rate between 125 to approximately 150-160 bpm; however, the highest desired test heart rate range is age-determined.

A valid test requires that the subject worked six minutes at a constant work rate to achieve a steady state heart rate (last two heart rate entries $\pm$ 4 bpm). The fitness test requires a minimum of six minutes and up to a maximum of ten minutes. The rider's heart rate during the final two minutes of pedaling is computed, using an equation that approximates the Astrand-Rhyming nomogram (104), to achieve a relative score that is measured in milliliters of oxygen per kilogram of body weight per minute (ml/kg/min). The resulting score, combined with values based on age and gender, places the individual in one of six fitness levels (**Table 1**). Category I being the lowest fitness level and Category VI the highest.

Fitness improvement program (GET FIT). Each member that falls into Category I must attend a mandatory exercise program (GET FIT). This program is designed to provide supervised guidance based on the quantity and quality of training

Table 1
Cycle ergometry **category I** parameters (minimal acceptable levels) using estimated maximum aerobic capacity

Age(y)	Category I	
	Male	**Female**
<29	<28[a]	<26
30-39	<27	<24
40-49	<25	<23
50>	<22	<20

[a]Estimated VO_{2max}, (ml O_2/kg/min) (Human Systems Center, Brooks AFB, TX)

recommended by the American College of Sports Medicine for developing and maintaining cardiorespiratory fitness (98). Members must participate in the GET FIT program for a minimum of 90 days. A 25% improvement in VO_{2max} is required to show satisfactory progress over the 90 day period. This improvement can occur through an absolute increase in aerobic capacity (VO_{2max} expressed in L/min), through a loss of body weight (since Category I standards are expressed relative to body weight), or a combination of both.

RECRUITMENT:

Subject selection. All active duty Air Force men and women who tested as a Category I, using cycle ergometry measures and estimated maximum oxygen consumption, were invited to participate. Participants were preliminarily screened by the fitness monitor prior to engaging in the test. The Monarch 818E cycle ergometer was utilized to estimate aerobic capacity and categorize fitness at two time points, the beginning and end of the 90-day program. All tests were considered valid and were conducted by trained and certified fitness monitors at McClellan Air Force Base.

Subjects were recruited in person at the entrance of the mandatory 90-day supervised fitness improvement program. Thirty-eight individuals volunteered to participate in the study. All participants were required to sign a consent to participate which was approved by the Human Subjects Review Committee (HSRC), University of California, Davis (145), and complete a *Health Risk Appraisal Questionnaire* (**Appendix A**). The questionnaire and individual medical records were reviewed to screen for eligible subjects. Individuals with known diabetes, pathologies of the vascular system, or those taking medications that might alter plasma lipids or cardiac function were excluded from the study (**Table 2**). Six individuals were excluded during the initial screenig process. The remaining thirty-two subjects were randomly assigned to two groups (**Table 3**). All engaged in similar modes of exercise training, and fifteen individuals

Table 2
Subject exclusion criteria[a]

Fitness category:	II III IV V VI
History of:	•Diabetes •Cardiovascular disease
Medications altering:	•Plasma lipoproteins •Cardiac function
Diet:	Any individual actively following a prescribed therapeutic diet.

[a]Subjects were interviewed by a health care professional and their medical records reviewed prior to acceptance into the study.

Table 3
Experimetal design and treatment intervention for the control and treatment groups

Subjects (n=32)	**Control (n=17)**	**Treatment (n=15)**
Lipid Profile		
•baseline	X	X
•post treatment	X	X
Food Frequency		
•baseline	X	X
•post treatment	X	X
Fitness Improvement Program (12 weeks)	X	X
Dietary Intervention (weekly)		X
Food Intake Records		X
Cycle Ergometry		
•baseline	X	X
•post treatment	X	X

(treatment group), received weekly individualized dietary counseling using the Food Guide Pyramid as an educational tool.

INTERVENTION:

Exercise training. All subjects participated in the mandatory exercise program from 6:00-7:00 a.m. three times a week. The mode of exercise incorporated the use of large muscle groups, and continued at an intensity of 60-80 percent of maximum heart rate for a duration of 40 minutes. Activities included walking, jogging, cycling and step-aerobic programs. Warm-up, cool-down and flexibility exercises were included within the hour. Participants were reevaluated at the end of the 90-day conditioning program using the cycle ergometry method of submaximal testing.

Dietary intervention. Both the control group and the treatment group completed a Food Frequency Questionnaire (**Appendix B**) at baseline and post treatment that was an expanded version of a form found in the Dairy Council pamphlet, "Food for a Healthy Heart" (146). This instrument was previously validated for calculating daily dietary fat and cholesterol intake.

The treatment group received weekly individualized dietary counseling by a Registered Dietitian. Six different topics were the focus of dietary education for the first half of the treatment period (**Table 4**). The same topics were repeated during the final six weeks of the program and the client was encouraged to instruct the dietitian using knowledge gained in previous instruction. This helped the dietitian assess the client's dietary knowledge and target areas in need of further education. Family members were encouraged to attend all instruction. The emphasis of education was on choosing a diet with plenty of vegetables, fruits and whole grains. Clients were encouraged to continue to

Table 4
Dietary counseling lesson plan for weeks 1-6, then repeated for weeks 7-12.

Session I	• Introduction to the US Dietary Guidelines • Understanding the Pyramid • The basics on keeping accurate food records
Session II	• Label reading techniques • The power of the Pyramid
Session III	• Where's the fat? • Trading down the fat scale
Session IV	• Where's the fiber? • Choosing whole foods and whole grains
Session V	• Food preparation tips • Modifying recipes
Session VI	• Dining out/special occasions • Coping skills/developing a strategy

consume favorite foods in moderation, considering no single food as inherently "bad" or "good" by itself. Balancing choices was emphasized. For example, if one chose to drink whole milk instead of skim, they could omit 2 teaspoons of fat elsewhere from their meal plan. Other approaches included eating smaller portions of high fat foods, using leaner methods in food preparation and trading down the fat scale to a lower fat alternative, for example, choosing nonfat frozen yogurt or ice milk instead of a premium ice cream. Whole foods and whole grains were encouraged as rich sources of dietary fiber. For instance, consuming whole fruit rather than juice was encouraged as was choosing brown rice rather than refined, white rice.

The *Food Guide Pyramid* was used as an educational tool. Participants and family members were taught to use the Food Guide Pyramid to modify food choices and dietary intake. Participants were required to keep three-day food intake records weekly for the duration of the treatment period. Instruction was given using food replicas (NASCO, Modesto CA) and standard measuring tools to ensure accuracy in record keeping and gauge progress in achieving the goals of the Food Guide Pyramid.

DATA COLLECTION:

Anthropometric data. All subjects were weighed and their height measured at the beginning and completion of the program. Body mass index (BMI) was calculated at these time points as an indicator of body composition and health outcomes. Desirable BMI ranges are 21.9 to 22.4 kg/m^2 for men and 21.3 to 22.1 kg/m^2 for women. Values greater than 27.8 kg/m^2 for men and 27.3 kg/m^2 for women are related to excess weight and increased risk of cardiovascular disease, high blood pressure and diabetes (147).

Dietary analysis. Food intake was analyzed using Nutritionist III software (version 7.2, 1991, N-Square Computing, Silverton, Ore). If a reported food was not in the database, the most similar food in the database was used. A few foods were added to the database according to information from the manufacturer of the product. Food serving sizes were manually converted to serving equivalents of the Food Guide Pyramid. These data were used to monitor compliance and assess changes in diet over time.

Plasma lipoproteins. To track changes in plasma lipoproteins, blood was drawn at two time points: at the entrance and completion of the 90-day program. At these time points, approximately 30 ml of blood was drawn by a certified laboratory technician following an overnight fast of 12-14 hours. Laboratory analysis of the blood samples was performed at the McClellan Clinic laboratory using Kodak Ektachem Clinical Chemistry Products and the Kodak Ektachem 700 Analyzer (Eastman Kodak

Co., Rochester, NY). The Kodak Ektachem Clinical Chemistry Slides CHOL (CAT 1688290) and TRIG (CAT 1648088) were used with the KODAK EKTACHEM HDL Cholesterol Kit to provide the completed lipoprotein profiles. LDL cholesterol and VLDL cholesterol were calculated from the results of these tests. The formulas used for calculation are:

Conventional (mg/dl)	***SI (mmol/L)***
VLDL = TRIG/5	VLDL = TRIG/2.2

LDL = CHOL-HDLC-VLDL

STATISTICAL ANALYSIS:

Data are expressed as mean ± standard deviation. Lipoprotein, anthropometric and fitness data were analyzed using two factor analysis of variance to identify interaction between gender and treatment using Statview 512+™ (Abacus Concepts, Inc., Berkeley, CA). One-factor analysis of variance was used to analyze changes in dietary consumption between gender within the treatment group. A two-tailed, paired "t" test was used to assess change in food group servings, food group fat, and dietary composition from baseline values for both genders. A Dunnett "t" test was used to compare the response of the treatment group to that of the control group when F values were significant at $p<0.05$.

RESULTS

Anthropometrics. Weight loss for the treatment group was significant (p=0.0001) after 90 days of dietary intervention plus exercise. No net weight loss was reported for the exercise only group. Within the treatment group, mean weight loss for women was less than for men, 1.37 ± 1.57 kg and 2.18 ± 1.15 kg, respectively (**Appendix C**); however, this gender difference was not significant. Reduction in BMI was significantly greater (p=0.0001) for the treatment group versus the controls (**Table 5**).

Food frequency data. Analysis of the food frequency data revealed the total fat intake for the control group remained constant throughout the study period, whereas a 39% reduction in fat was achieved for the subjects who received dietary intervention (**Table 6**). A similar reduction in fat (44%) was seen for this group using 3-day food intake record and Nutritionist III software analysis. These results suggest the food frequency questionnaire provided relatively accurate information for calculating dietary fat intake and assessing general trends in diet over time.

Diet composition. A shift in energy from fat (39% to 23%) to carbohydrates (44% to 58%) was observed (**Figure 1**). In addition, saturated fat decreased from 14% to 6%. Total energy intake decreased for men and women by a mean of 233 kcals (p=0.007) and 101 kcals (p=0.05), respectively (**Table 7**). Mean dietary cholesterol intake decreased substantially, with greater

Table 5
Characteristics of subjects

Parameter	Control (n=17)	Treatment (n=15)
Gender		
Men	12	8
Women	5	7
Age(y)	32.7±7.0[a]	33.3 ± 6.0
Weight(kg)		
Baseline	75.5 ± 9.4	77.2 ± 13.5
Post treatment	75.7 ± 9.3	75.4 ± 13.1[b]***
Body mass index[c]		
Baseline	25.1± 2.4	25.6 ± 2.8
Post treatment	25.2 ± 2.3	25.1 ± 2.9[b]***

[a]Mean ± standard deviation.
[b]Response of treatment vs. control group to intervention.
[c]Body mass index (kg/m^2)
***P<.001

Table 6
Changes in total daily dietary fat intake using food frequency data

Time point	Fat	
	Control (g/d)	Treatment (g/d)
Baseline	109 ± 22[a]	97 ± 17
Post	110 ± 24	59 ± 14[b]***

[a]Mean ± standard deviation.

[b]Response of treatment vs. control group to intervention.

*** P<.001

Figure 1. Changes in diet composition for the treatment group from baseline expressed in percentage of energy intake.

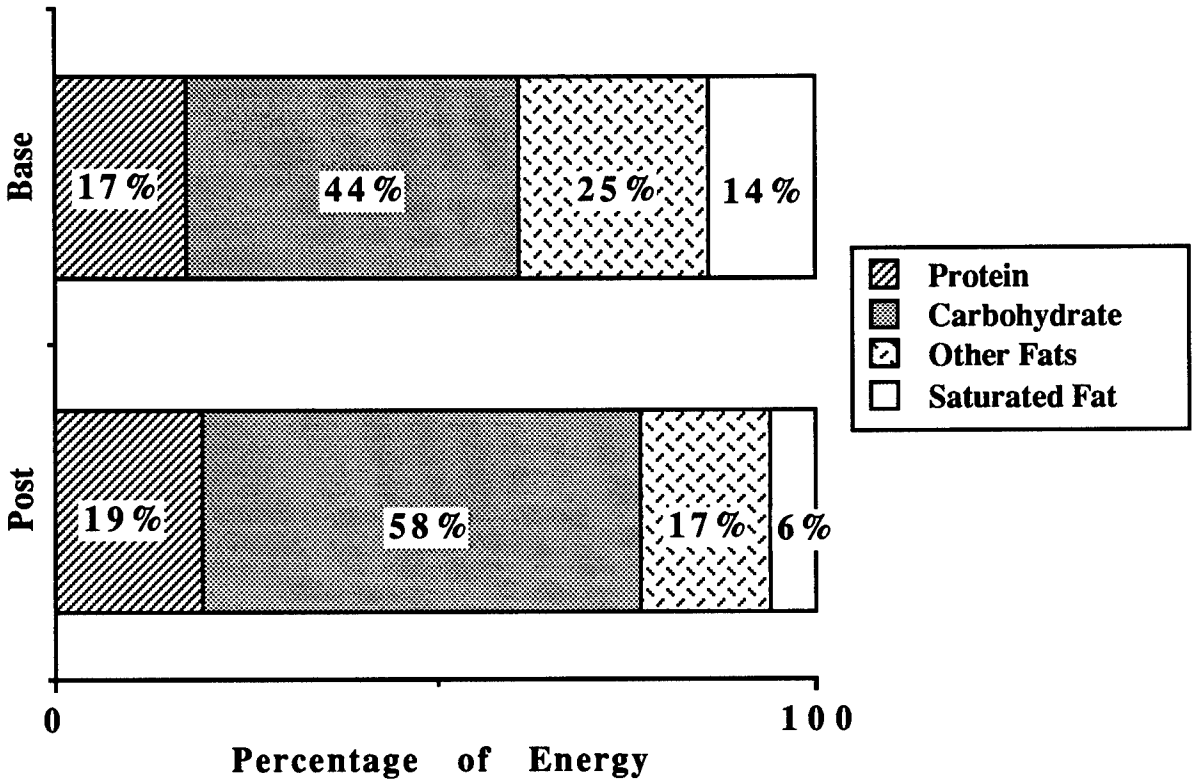

Base
17 %
44 %
25 %
14 %
Post
19 %
58 %
17 %
6 %
Protein
Carbohydrate
Other Fats
Saturated Fat
0
100
Percentage of Energy

Table 7
Changes in energy composition, dietary cholesterol and fiber intake by gender for treatment group

Parameter	Pre treatment		Post treatment[a]	
	male (n=8)	female (n=7)	male (n=8)	female (n=7)
Energy(kcal/d)	2347 ± 278[b]	2083 ± 256	2114 ± 210[c**]	1982 ± 114
Protein(%)	17 ± 1	15 ± 3	19 ± 2	19 ± 5
Carbohydrate(%)	45 ± 5	44 ± 8	60 ± 4[c**]	55 ± 4[d**]
Total fat(%)	38 ± 4	41 ± 7	21 ± 3[c***]	26 ± 3[d***]
Saturated fat(%)	14 ± 3	13 ± 2	6 ± 2[c***]	7 ± 2[d**]
Cholesterol(mg/d)	482 ± 188	462 ± 161	188 ± 47[c**]	222 ± 76[d*]
Fiber(g/1000 kcal/d)	5 ± 3	4 ± 3	8 ± 2	10 ± 4[d**]

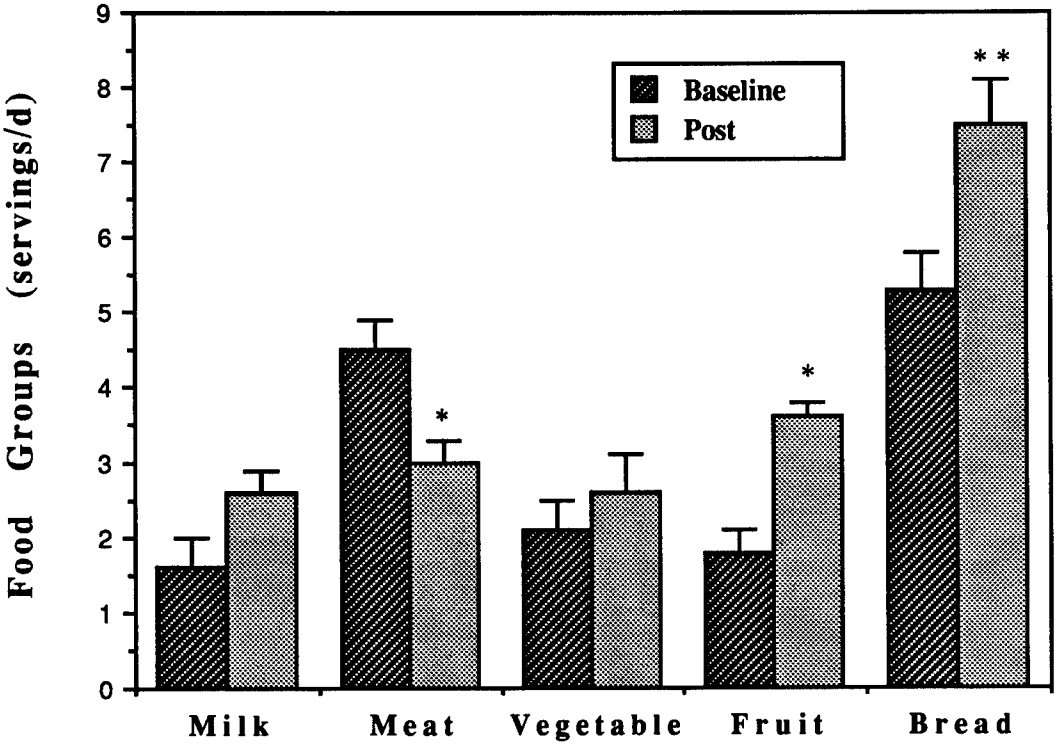
[a]Values for gender determined using three-day food records from week 12 of the treatment period.
[b]Mean ± standard deviation
[c]Post treatment values for males; significance compared to baseline.
[d]Post treatment values for females; significance compared to baseline.
* P<.05
** P<.01
*** P<.001

declines observed for men (-294 mg/d) than women (-240 mg/dl), although this gender difference was not significant.

Food category sevings. By the end of the treatment period, marked improvements in dietary choices were observed for the treatment group (**Figure 2**). These changes reflect a shift toward an increase in daily servings of *milk* (1.6 to 2.6), *fruit* (0.8 to 3.6), *vegetables* (2.0 to 2.6), and *bread* (5.3 to 7.5), and a reduction in *meat* servings (4.5 to 3.0). **Table 8** identifies mean changes in food group servings by gender for the treatment group. Both men (p=0.006) and women (p=0.001) had significant increases in daily fruit consumption. Additionally, a significant reduction in meat (p=0.007) and increase in bread servings (p=0.007) was observed for men. The meat and "*others*" category contributed the greatest amount of fat at baseline for both genders (**Figure 3**). The largest percent decrease in fat for both men (p=0.007) and women (p=0.03) came from the *meat* group (49% and 41%) The *"others"* category decreased 25% and 32% respectively, but this was not significant (**Table 9**). Mean post treatment dietary profiles satisfied at least the minimum number of recommended servings for each food group.

Dietary fiber. A 71% increase (p=0.007) in total daily grams of dietary fiber was achieved from baseline for the treatment group (**Figure 4**). When expressed per 1000 kcals, an 84% increase was observed. At the end of the treatment period, a significantly greater (p=0.05) fiber intake (mean=24 g/d) was found in subjects who consumed less than 20% of their total energy intake as fat than in those those consuming greater than 20% fat calories (mean =17 g/d).

Figure 2. Changes in mean food group servings for the treatment group expressed in number of servings/d.



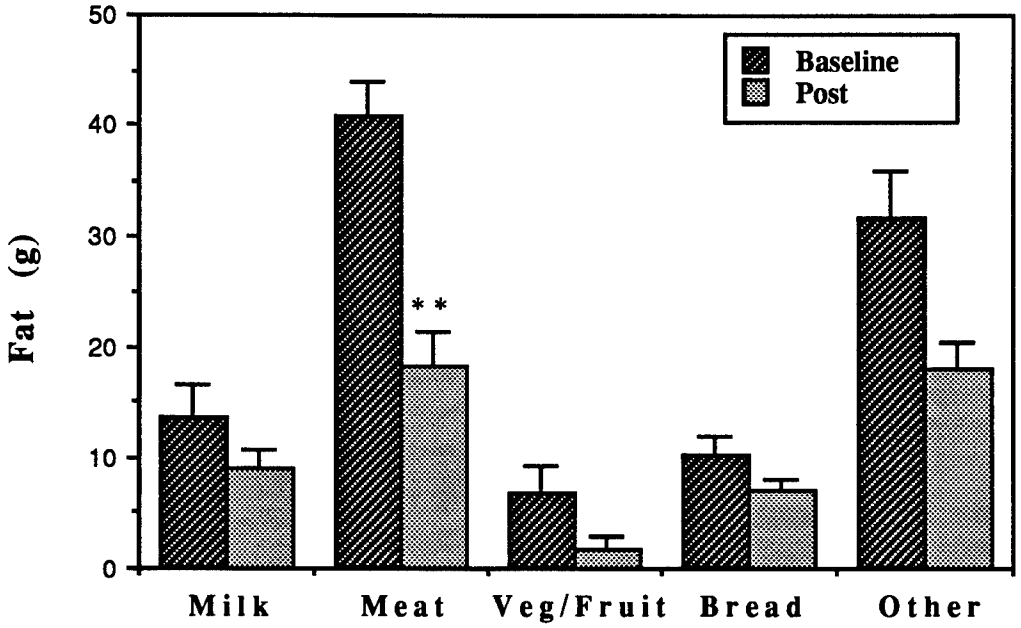
* Significant at P<0.05
** Significant at P<0.01

Table 8
Changes in food group servings by gender for treatment group

Food group	Pre treatment		Post treatment	
	male (n=8)	female (n=7)	male (n=8)	female (n=7)
Milk(servings/d)	2.0 ± 1.5[a]	1.1 ± 1.3	2.6 ± 1.4	2.5 ± 1.3
Meat(servings/d)	4.7 ± 1.8	4.2 ± 1.6	2.9 ± 0.8[b**]	3.0 ± 1.7
Vegetable(servings/d)	2.3 ± 1.8	1.9 ± 1.2	2.6 ± 1.7	2.6 ± 2.0
Fruit(servings/d)	0.9 ± 1.1	0.6 ± 1.1	3.4 ± 1.1[b**]	3.9 ± 0.7[c**]
Bread(servings/d)	6.3 ± 2.1	4.1 ± 1.6	8.9 ± 1.2[b**]	5.9 ± 2.2

[a]Mean ± standard deviation.
[b]Post treatment values for males; significance compared to baseline.
[c]Post treatment values for females; significance compared to baseline.
** P<.01

Figure 3. Changes in mean total fat intake per food group for the treatment group expressed in g/d.



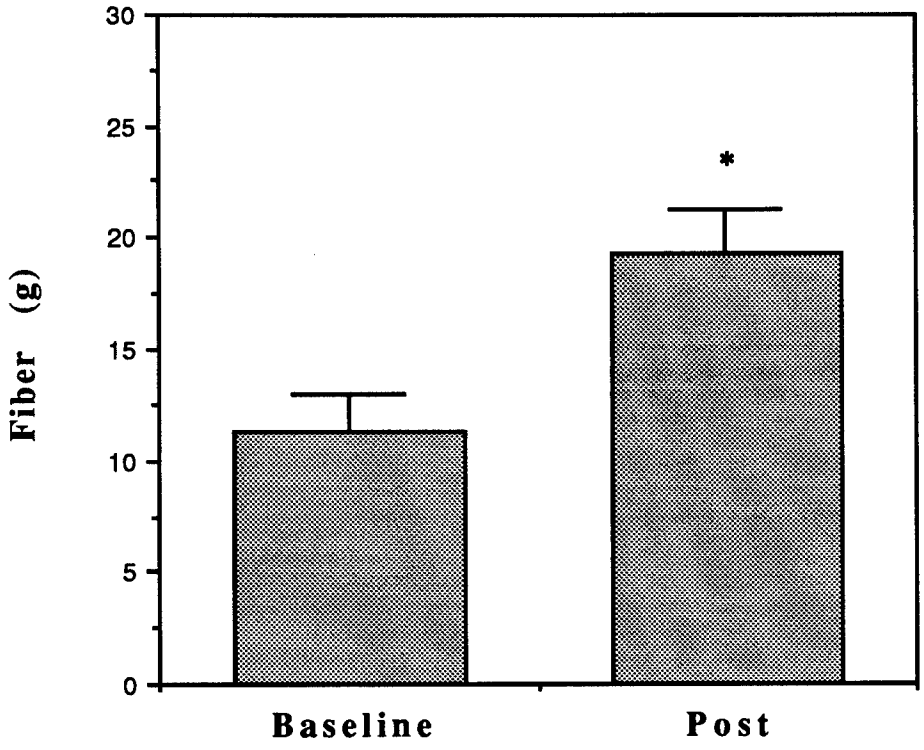
** Significant at P<0.01

Table 9
Total daily grams of fat from each food group per person

Food group	Pre treatment		Post treatment	
	male (n=8)	female (n=7)	male (n=8)	female (n=7)
Milk(fat,g)	13.2 ± 9.4[a]	14.2 ± 14.9	7.1 ± 7.2	11.0 ± 7.6
Meat(fat,g)	44.3 ± 8.9	36.7 ± 19.8	17.5 ± 7.0[b]**	18.7 ± 17.2[c]*
Vegetable(fat,g)	6.1 ± 5.4	7.9 ± 12.2	3.0 ± 5.6	0.4 ± 1.3
Fruit(fat,g)	NA[d]	NA	NA	NA
Bread(fat,g)	12.9 ± 7.7	7.1 ± 3.8	8.3 ± 4.0	5.8 ± 2.5
"Others"(fat,g)	27.5 ± 12.1	36.7 ± 19.8	14.1 ± 8.4	22.4 ± 9.1

[a]Mean ± standard deviation.
[b]Post treatment values for males; significance compared to baseline.
[c]Post treatment values for females; significance compared to baseline.
[d]Not applicable.
* P<.05
** P<.01

Figure 4. Changes in mean total daily dietary fiber intake for the treatment group expressed in g/d.



* Significant at P<0.01

Plasma lipoproteins. A 9% reduction (p=.002) in total cholesterol and 13% reduction (p=0.002) in LDL-C was achieved in the treatment group while the control group experienced no change in these measurements (**Table 10**). No significant change in HDL-C or TG concentration was observed for either group. A significant difference due to gender was found in the treatment group at baseline for HDL (p=0.008) and TG (p=0.05) (**Table 11**). HDL-C was significantly higher and TG concentration significantly lower in females than males in the treatment group. However, this difference was not significant at the post treatment measurement.

Fitness. Although both groups had improvements in estimated maximum VO_2 values (control=14%, treatment=38%) because of the exercise training, this increase was significantly greater for the diet plus exercise group than the exercise only group (p=0.0001) (**Figure 5**). Within the treatment group, women achieved improvements in aerobic capacity of 55% versus 28% for men which were significantly different (p=0.0003) (**Table 12**).

Table 10
Changes in fasting plasma lipoproteins between groups

Parameter	Control (n=17)		Treatment (n=15)	
	Pre	Post	Pre	Post
Total Cholesterol(mmol/L)[a]	4.87 ± 0.89[b]	4.85 ± 0.88	4.96 ± 1.00	4.48 ± 0.89[c]**
LDL Cholesterol(mmol/L)[a]	3.03 ± 0.81	3.01 ± 0.88	3.04 ± 0.84	2.66 ± 0.72[c]**
HDL Cholesterol(mmol/L)[a]	1.21 ± 0.21	1.38 ± 0.69	1.33 ± 0.49	1.22 ± 0.34
Triglycerides(mmol/L)[d]	1.40 ± 0.48	1.36 ± 0.43	1.40 ± 0.53	1.52 ± 0.64

[a]To convert mmol/L cholesterol or lipoprotein cholesterol to mg/dL, multiply mmol/L by 38.7. To convert mg/dL cholesterol to mmol/L, multiply mg/dL by 0.026. Cholesterol of 5.00 mmol/L = 193 mg/dL.

[b]Mean ± standard deviation.

[c]Response of treatment vs. control group to intervention.

[d]To convert mmol/L triglyceride to mg/dl, multiply mmol/L by 88.6. To convert mg/dL triglyceride to mmol/L, multiply mg/dL by 0.0113. Triglycerides of 1.80 mmol/L = 159 mg/dL.

** P<.01

Table 11
Fasting plasma lipoprotein values at baseline by group and gender

Parameter	Control		Treatment	
	male (n=12)	female (n=5)	male (n=8)	female (n=7)
Total cholesterol(mmol/L)[a]	4.81± 0.78[b]	5.01 ± 1.22	5.05 ± 0.75	4.85 ± 1.29
LDL cholesterol(mmol/L)[a]	3.02 ± 0.74	3.06 ± 1.05	3.15 ± 0.65	2.91 ± 1.06
HDL cholesterol(mmol/L)[a]	1.15 ± 0.22	1.35 ± 0.10	1.10 ± 0.23	1.60 ± 0.60[c]**
Triglycerides(mmol/L)[d]	1.40 ± 0.43	1.42 ± 0.63	1.73 ± 0.47	1.02 ± 0.29[c]*

[a]To convert mmol/L cholesterol or lipoprotein cholesterol to mg/dL, multiply mmol/L by 38.7. To convert mg/dL cholesterol to mmol/L, multiply mg/dL by 0.026. Cholesterol of 5.00 mmol/L = 193 mg/dL.

[b]Mean ± standard deviation.

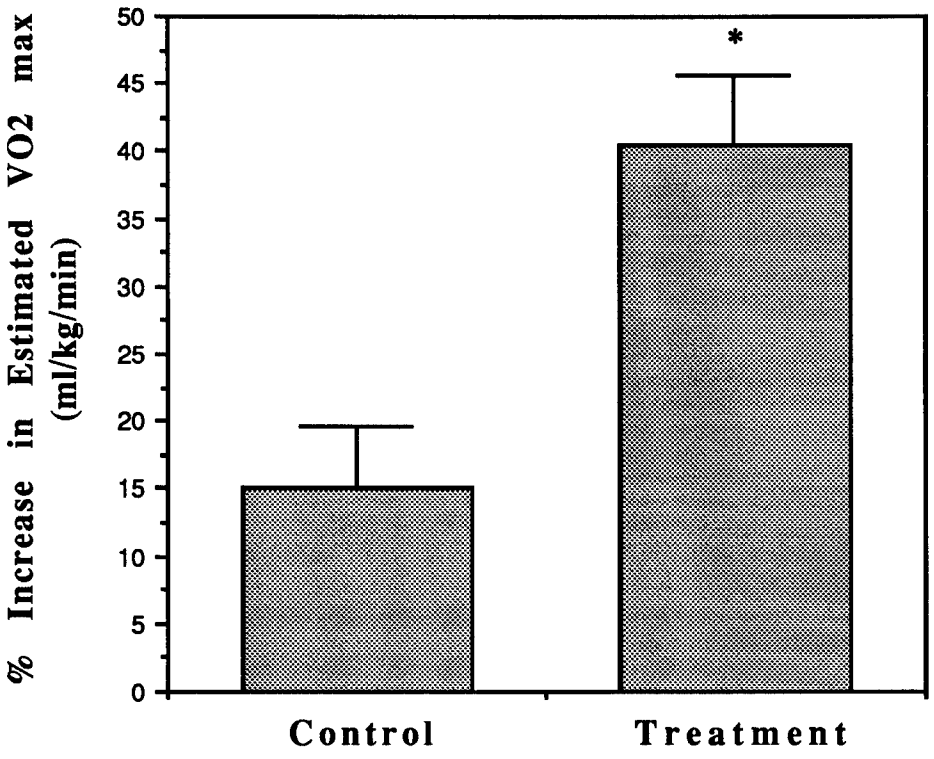
[c]Baseline values for females vs. males in treatment group.

[d]To convert mmol/L triglyceride to mg/dl, multiply mmol/L by 88.6. To convert mg/dL triglyceride to mmol/L, multiply mg/dL by 0.0113. Triglycerides of 1.80 mmol/L = 159 mg/dL.

*P<.05

**P<.01

Figure 5. Percent increase in mean estimated VO_{2max} for the control and treatment groups after twelve weeks.



* Significant at P<0.01

Table 12
Change in aerobic capacity for the treatment group by gender

Parameter	Pre treatment male (n=8)	Pre treatment female (n=7)	Post treatment male (n=8)	Post treatment female (n=7)
Estimated VO_{2max}[a]	25.31 ± 3.24[b]	22.79 ± 2.60	33.09 ± 5.87**	36.06 ± 3.10**
Estimated VO_{2max}[c]	2.19 ± 0.32	1.50 ± 0.12	2.80 ± 0.60**	2.32 ± 0.12***

[a]Estimated VO_{2max} (ml/kg/min)
[b]Mean ± standard deviation.
[c]Estimated VO_{2max} (L O_2/min)
** P<.01
*** P<.001

DISCUSSION

The *Second Adult Treatment Panel II report* (5) suggests a public health approach to lower blood cholesterol levels in the American population. One method is to shift the distribution of cholesterol levels in the entire population to a lower range through dietary modification. An increased emphasis on physical activity and weight loss is recommended as a component of diet therapy. The general aim of diet therapy is to reduce serum cholesterol while maintaining a nutritionally adequate eating plan (5).

In this study, we found the Food Guide Pyramid to be an effective tool in achieving a nutritionally adequate, yet cardioprotective diet. We observed a significant diet effect on the reduction in CHD risk through the lowering of TC (9%), LDL-C (14%), BMI (2%), and improvement in aerobic capacity. A substantial reduction in total dietary fat (39% to 23%) and saturated fat (14% to 6%) was achieved. This decrease in fat energy was replaced with a larger percentage of energy from carbohydrate (45 to 58%). Dietary intake of cholesterol decreased in concert with the consumption of animal products. The overall shift in diet pattern mirrored the NCEP Step II guidelines that recommends a total fat intake <30% of calories, a saturated fat intake <7%, and a dietary cholesterol intake of <200 mg/d. It is well established that adherence to such a regimen results in reductions in total cholesterol of 10% to 20%, and the addition of

soluble fiber may lead to further reductions in TC of 1% to 10% (148).

The observed weight loss of 1.8 kg for the treatment group may be related to the unintentional mean daily reduction in energy intake of 172 kcals in combination with the 41% reduction in total fat consumption. These findings are consistent with the literature that weight loss is associated with low fat diets even when isocaloric intakes are reported. For example, women in the Women's Health Trial consumed an average of 22% of energy from fat and lost an average of 3 kg after 1 year without trying to lose weight or restrict energy intake (149). The exercise only group experienced no change in body weight. It is possible that they had an increase in muscle to fat ratio, and thus no net weight loss. However, it is more likely they had a compensatory increase in energy intake to balance expenditure, which supports the view that weight loss due to exercise alone will be minimal compared to a combination of exercise with dietary modification (150).

Despite the exposure of the Food Guide Pyramid throughout the military installation, we observed no evidence of change in the control group for total fat intake using food frequency data, anthropometric or biochemical values. Although the validity and the reliability of the food frequency method is open to question, it is found to be a valid tool for determining habitual intake (151) and is considered appropriate for classifying subjects according to their fat consumption (152). It appears that the availability of nutrition information alone may not be enough to effect change in behavior (11, 153). Conversely, when dietary education was

provided to a subset of the population, significant changes in food intake, BMI, TC and LDL-C occurred.

The largest reduction in fat for our study as well as in other low fat dietary interventions, was from fats/oils, red meats and dairy products (154-156). Subjects commented that the easiest changes to make were in the fats/oils group due to the availability of acceptable reduced fat and fat free alternatives. Other investigators have reported similar findings (156, 157).

As the consumption of fruits, vegetables and whole-grains increased, so did dietary fiber. Incorporation of carbohydrate-rich, high fiber foods improved the ratio of dietary fiber to energy intake. We had no reports of subjects feeling hungry or deprived throughout the study period. Several participants commented on the difficulty in consuming the larger quantities of food. The added food bulk may have contributed to satiety. Other researchers have found that when high fiber food constitutes a major share of the caloric intake, it limits the capacity to eat other foods (158, 159). In our study, we observed that individuals with the highest dietary fiber intake (24 g/d) had the lowest intake of fat (<20%).

To our knowledge, this study is one of the first to examine the effects of diet on the response to exercise training. The large difference in fitness improvement between the diet plus exercise group (38%) and the exercise only group (14%) was an unexpected finding. Exercise prescriptions of similar frequency, intensity, duration of training and mode of activity have achieved increases in VO_{2max} of 15% to 30%. Changes in VO_{2max} greater than 30% are

usually associated with large total body mass and fat weight loss, or in persons with very low initial levels of fitness. For example, data from training studies using subjects with varied levels of VO_{2max}, total body mass and fat weight indicate that changes occur in relation to their initial values (160-163). The lower the initial VO_{2max}, the larger the percentage of improvement found and the higher the percent body fat, the bigger the reduction. The greater improvements for the women in our study may be partly attributed to their lower initial levels of fitness and potentially greater loss of fat compared to muscle weight. Information is not available on body composition to fully assess fat loss.

Furthermore, it is quite possible that the weekly contact with the same dietitian and the individual attention received by the treatment group enhanced the treatment subjects' motivation to engage in additional exercise outside of the program. Observed changes in body weight and a perceived sense of control over one's eating behavior may have further motivated some to exercise at a greater intensity or frequency. These factors argue for the importance of personal contact to provide education for behavioral modification rather than simply providing information.

One observed limitation is that exercise records were not incorporated into the study design. It was assumed all subjects would engage in similar supervised and leisure-time activities. Furthermore, we chose to limit the focus on exercise patterns to lessen any individual change in exercise behavior. It is of interest to note that the treatment group also increased their overall carbohydrate intake by 10 percent. Carbohydrates are broken

down into glucose and stored in muscle as glycogen. Increased carbohydrate in the diet results in greater glycogen stores, which may affect physical performance. Further research is needed to quantify the absolute effect of carbohydrate intake on submaximal fitness measurements.

Physical training did not increase HDL-C as reported by others (142, 164). However, other investigators have failed to demonstrate effects of exercise on HDL (127-132). This lack of effect may be attributed to a level of exercise below a determined threshold for change (126), the relatively short study period, or the relatively high carbohydrate intake from baseline (165).

The results of this study are of considerable public health importance. Free-living, sedentary men and women made substantial changes in diet through simple modifications to include a minimum number of healthful selections from the major food groups. This positive approach to eating in combination with exercise produced weight loss, a reduction in CHD risk and an enhanced cardiorespiratory response to exercise training. Dietary education emerged as an important vehicle to facilitate change in behavior. Future intervention programs should include a prescription of sensible eating and moderate exercise to maximize the reduction in chronic disease and contribute to the health of the Nation.

SUMMARY

This study illuminates the profound impact of dietary modification combined with moderate exercise on plasma lipoproteins, fitness improvement and reduction in cardiovascular risk in healthy adults.

The Food Guide Pyramid emerged as a highly effective educational tool to communicate the Dietary Guidelines for Americans. The power of the Pyramid was maximized when linked with individual dietary counseling. When applied in this setting, the educator facilitated in adapting the Pyramid to a variety of dietary behaviors, food preferences and lifestyles. By using a goal oriented, client centered, positive approach to change, a therapeutic diet similar to the ***NCEP Step-2*** was achieved. Additionally, secondary improvements in lipoprotein, anthropometric, and fitness values were realized.

With a focus toward the future, and a vision to optimize the health of the Nation by the year 2000, nutrition intervention must be proactive, adaptive and user friendly. Our findings suggest the combined effect of diet and exercise is additive in reducing CHD risk. We conclude a prescription of sensible eating and moderate exercise is a cost-effective intervention with tremendous potential to limit disease and optimize health for the Nation.

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APPENDIX A

HUMAN CONSENT FORM

HEALTH RISK ASSESSMENT QUESTIONNAIRE

Consent to Participate in a Research Study
University of California, Davis

Changes in Plasma Lipoproteins with Improved Fitness and Individualized Dietary Modification in Active Duty Air Force Members Enrolled in the GET-FIT Program.

Investigators:
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Purpose

You are invited to participate in a research project to study the effects of fitness and diet on plasma lipoproteins and reduction in heart disease risk. As a result of the study, we hope to learn if improved fitness and modification in diet can improve known health risks associated with coronary heart disease.

Procedures

If you are interested in participating in this study, you will be asked to donate blood twice over a three month period. Each time 30 ml of blood (roughly 2 tablespoons) will be taken. The blood draws will be requested by a provider at McClellan clinic and will require an overnight fast of 12-14 hours. You will also be asked to complete AFLC/AFSC Form 49, Health Risk Appraisal Questionnaire, at the entrance and completion of the program.

Risks

There is some degree of risk associated with venipuncture required to draw blood. Venipuncture may result in a slight risk of infection, bruising, and/or discomfort from the insertion of the needle. This risk is minimal, however.

Benefits

To You: You will gain a greater understanding of how fitness and diet affects your plasma lipoprotein particles. You may or may not receive weekly dietary counseling by a Registered Dietitian to help you individualize a health eating plan. There will be no direct benefit to you as a result of this research other than the benefits received by engaging in the program.

To the Air Force: We will gain a better understanding of the role of fitness and diet in reducing health risk in active duty personnel. Knowledge gained may be used to improve certain health promotion programs to enhance combat readiness.

Subject's Initials_____

Alternative Treatment

The alternative is not to participate in this study. If you choose not to participate, you will still be required to participate in the mandatory 90-day GET-FIT program as directed by the Air Force.

Confidentiality

Every effort will be made to keep the names of the participants confidential. Any publications resulting from this study will have the names of the participants represented by a number so as to prevent any identifiable patient name being associated with a particular result. However, absolute confidentiality can not be guaranteed, since research documents are not protected from supoena.

Cost/Compensation

There will be no compensation for participation in the study. In accordance with federal regulations, we are obliged to inform you about the Medical Center's policy in the event of physical injury. If you are physically injured as a direct result of research procedures not done primarily for your own benefit, undertaken at the University of California facilities, you will receive medical treatment at no cost. The University of California does not provide any other compensation for injury.

Right to Refuse or Withdraw

You are under no obligation to participate in this study. Furthermore, if you begin the study, you may decide to withdraw at any time without prejudice. You may refuse to participate and still receive the care you would normally receive if you were not in the study.

Questions

You will be given a signed and dated copy of this form to keep. You will also be given a copy of the experimental subjects Bill of Rights. If you have any questions, please feel free to call me.

Patricia J. Gambera, Capt, USAF,BSC (916) 729-7096

Your signature below, indicates that you have decided to volunteer as a research subject and that you read and understand the information provided above, and the Experimental Subject Bill of Rights.

_____ _____

Date Signature of participant or legal representative

_____ _____

Date Signature of participant of legal representative

HEALTH RISK APPRAISAL QUESTIONNAIRE

NAME _____

UNIT _____

PHONE _____

(This Form is Subject to the Privacy Act of 1974 - Use Blanket PAS DD FORM 2005)

Directions

Your answers will be treated as confidential, only group information would be available to the commander. Please keep the coupon with your participant number on it. You may need it to claim your computer report. To get the most accurate results answer as many questions as you can. If you do not know the answer leave it blank. Questions with a ★ (star symbol) are important to your health, but are not used by the computer to calculate your risk. However, your answers may be helpful in planning your health and fitness program.

- USE A NO. 2 PENCIL ONLY.
- FILL THE OVAL COMPLETELY. → ● ●
- WRITE THE NUMBER IN THE BOX PROVIDED, THEN MARK THE CORRESPONDING OVAL TO THE RIGHT.
- ERASE CHANGES COMPLETELY.
- MAKE NO STRAY MARKS ON THE FORM.

Marking Example
If your age is 29: [2][9]

MARK YOUR IDENTIFICATION NUMBER HERE → IDENTIFICATION NUMBER

THEN BEGIN THE QUESTIONNAIRE HERE

1. **Sex:** ○ Male ○ Female
2. **Age:** Years
3. **Height: (without shoes/no fractions)** Feet Inches — Ft. 1 2 3 4 5 6 7 — In. 1 2 3 4 5 6 7 8 9 10 11
4. **Weight: (without shoes/no fractions)** Pounds
5. **Body Frame Size:** ○ Small ○ Medium ○ Large
6. **Have you ever been told you have diabetes (or sugar diabetes)?** ○ Yes ○ No
7. **Are you now taking medicine for high blood pressure?** ○ Yes ○ No
8. **What is your blood pressure now?**
   - Systolic (High Number)
   - Diastolic (Low Number)
9. **If you do not know the numbers, mark the response that describes your blood pressure.** ○ High ○ Normal or Low ○ Don't Know
10. **What is your TOTAL cholesterol level (based on a blood test)?**
11. **What is your HDL Cholesterol (based on blood test)?**
12. **How many cigars do you usually smoke per day?**
13. **How many pipes of tobacco do you usually smoke per day?**
14. **How many times per day do you usually use smokeless tobacco? (chewing tobacco, snuff, pouches, etc.)**

019284

PLEASE DO NOT MARK IN THIS AREA

★ U.S. GOVERNMENT PRINTING OFFICE:1992—649-976

26. How long has it been since your last breast X-Ray? (Mammogram)

- ○ Less Than 1 Year Ago
- ○ 1 Year Ago
- ○ 2 Years Ago
- ○ 3 or More Years Ago
- ○ Never

27. How many women in your natural family (mother, and sisters only) have had breast cancer?

⓪ ① ② ③ ④ ⑤ ⑥ ⑦ ⑧ ⑨ ⑩ or more Women

28. Have you had a hysterectomy operation?

- ○ Yes
- ○ No
- ○ Not Sure

29. How long has it been since you had a pap smear test?

- ○ Less Than 1 Year Ago
- ○ 1 Year Ago
- ○ 2 Years Ago
- ○ 3 or More Years Ago
- ○ Never

★ 30. How often do you examine your breasts for lumps?

- ○ Monthly
- ○ Once Every Few Months
- ○ Rarely or Never

★ 31. About how long has it been since you had your breasts examined by a physician or nurse?

- ○ Less Than 1 Year Ago
- ○ 1 Year Ago
- ○ 2 Years Ago
- ○ 3 or More Years Ago
- ○ Never

★ 32. About how long has it been since you had a rectal exam?

- ○ Less Than 1 Year Ago
- ○ 1 Year Ago
- ○ 2 Years Ago
- ○ 3 or More Years Ago
- ○ Never

WOMEN GO TO QUESTION # 34

MEN

★ 33. About how long has it been since you had a rectal or prostate exam?

- ○ Less Than 1 Year Ago
- ○ 1 Year Ago
- ○ 2 Years Ago
- ○ 3 or More Years Ago
- ○ Never

★ 34. How many times in the last year did you witness or become involved in a violent fight or attack where there was a good chance of a serious injury to someone?

- ○ 4 or More Times
- ○ 2 or 3 Times
- ○ 1 Time or Never
- ○ Never

★ 35. Considering your age, how would you describe your overall physical health?

- ○ Excellent
- ○ Good
- ○ Fair
- ○ Poor

★ 36. In an average week, how many times do you engage in physical activity (exercise or work which lasts at least 20 minutes without stopping and which is hard enough to make you breathe heavier and your heart beat faster)?

- ○ Less Than 1 Time Per Week
- ○ 1 or 2 Times Per Week
- ○ At Least 3 Times Per Week

★ 37. If you ride a bicycle, motorcycle or all-terrain vehicle (ATV) what percent of the time do you wear a helmet?

- ○ 75% to 100%
- ○ 25% to 74%
- ○ Less Than 25%
- ○ Does Not Apply to Me

★ 38. Do you eat some food every day that is high in fiber, such as whole grain bread, cereal, fresh fruits or vegetables?

- ○ Yes
- ○ No

★ 39. Do you eat food every day that is high in cholesterol or fat, such as fatty meat, cheese, fried foods, or eggs?

- ○ Yes
- ○ No

APPENDIX B

FOOD FREQUENCY FORM

FOOD FREQUENCY FORM

WHAT DO YOU EAT ? IN THE COLUMN NEXT TO EACH FOOD, WRITE DOWN THE NUMBER OF SERVINGS YOU USUALLY EAT IN A DAY, AND OVER ONE WEEK. CONSIDER THE AMOUNT LISTED BY EACH FOOD AS 1 SERVING, EXCEPT IN THE MEAT GROUP. A *5 -OUNCE* PORTION, THE AMOUNT MOST ADULTS EAT COUNTS AS *2-SERVINGS* .

TO SCORE MIXED FOODS, SUCH AS CHEESEBURGERS, PIZZA, TACOS OR FROZEN DINNERS, COUNT THE INGREDIENTS SEPARATELY IN EACH FOOD GROUP. FOR EXAMPLE, COUNT A TACO AS A TACO SHELL, HAMBURGER MEAT, LETTUCE, TOMATO, AND CHEESE.

MILK AND MILK PRODUCTS

FOOD ITEM *AMOUNT = 1 SERVING*	*SERVINGS PER DAY*	*SERVINGS PER WEEK*
NONFAT MILK (1 CUP)		
NONFAT YOGURT (1 CUP)		
NONFAT FROZEN YOGURT (1 CUP)		
LOW FAT MILK (1%) ,(2%) CHOCOLATE (1 CUP)		
LOW FAT YOGURT, PLAIN OR WITH FRUIT (1 CUP)		
LOW FAT COTTAGE CHEESE (1 CUP)		
LOW FAT OR PART -SKIM CHEESE (1 OZ)		
FROZEN YOGURT, ICE MILK (1 CUP)		
WHOLE MILK (1 CUP)		
COTTAGE CHEESE, REGULAR (1 CUP)		
CHEESE (1 OZ)		
CREAM SOUP (1 CUP)		
MILKSHAKE (10 OZ)		
PUDDING (1 CUP)		
ICE CREAM (1 CUP)		
ICE CREAM , RICH (1 CUP)		
CUSTARD, BAKED (1 CUP)		

MEAT AND ALTERNATES

FOOD ITEM *2-3 OUNCES = 1 SERVING*	*SERVINGS PER DAY*	*SERVINGS PER WEEK*
DRIED PEAS & BEANS ,COOKED (1 CUP)		
SHRIMP, LOBSTER (3 OZ)		
SCALLOPS (3 OZ)		
TUNA, CANNED IN WATER (3 OZ)		
SOLE, BROILED OR BAKED (5 OZ)		
TOFU (1/2 CUP)		
BEANS, REFRIED OR BAKED (1 CUP)		
CRAB, CLAMS (3 OZ)		
TUNA, CANNED IN OIL (3 OZ)		
COD, TROUT, BROILED OR BAKED (5 OZ)		
CHICKEN, TURKEY, ROASTED, NO SKIN (5 OZ)		
SALMON, BROILED OR BAKED (5 OZ)		
FRIED FISH (5 OZ)		
CHICKEN WITH SKIN, ROASTED (5 OZ)		
EGGS (2)		
LEAN BEEF, VEAL, LAMB, HAM (5 OZ)		
BEEF LIVER, FRIED (5 OZ)		
FRIED CHICKEN WITH SKIN (5 OZ) 2 PIECES		
HAMBURGER, LEAN OR REGULAR (5 OZ)		
PORK ROAST (5 OZ)		
LUNCHEON MEAT (2 OZ)		
FRANKFURTERS (2)		
PORK OR LAMB CHOPS (5 OZ) 2 CHOPS		
PEANUT BUTTER (2 TB)		
NUTS (1/4 CUP)		

VEGETABLES AND FRUITS

FOOD ITEM *AMOUNT LISTED = 1SERVING*	*SERVINGS PER DAY*	*SERVINGS PER WEEK*
GREEN SALAD (1 CUP)		
VEGETABLES, RAW (1 CUP) OR COOKED (1/2 CUP)		
FRESH FRUIT (1 PIECE OR 1/2 CUP)		
DRIED FRUITS (1/4 CUP)		
POTATO, BAKED OR BOILED (1)		
TOMATO SAUCE (1/2 CUP)		
COLESLAW (1/2)		
VEGETABLE SOUP (1 CUP)		
HASH BROWN POTATOES (1 PATTY)		
FRENCH FRIES (1 ORDER)		
POTATO SALAD (3/4 CUP)		
AVOCADO (1/2)		

BREADS AND CEREALS

FOOD ITEM *AMOUNT LISTED = 1 SERVING*	*SERVINGS PER DAY*	*SERVINGS PER WEEK*
CEREAL, COOKED OR DRY (1 CUP)		
BREAD (1 SLICE)		
SODA OR WATER CRACKERS (5)		
ENGLISH MUFFIN, BAGEL (1/2)		
RICE OR PASTA, COOKED (1/2)		
CORN TORTILLA (1)		
HAMBURGER OR HOT DOG BUN (1/2)		
ROLL (1)		
MUFFIN,BISCUIT (1)		
FLOUR TORTILLA, TACO SHELL (1)		
CRACKERS (5)		
PANCAKES (2)		
WAFFLES, FROZEN (2 SQUARES)		
GRANOLA (1/2 CUP)		
CROISSANT (1)		

EXTRA FOODS

FOOD ITEM AMOUNT = 1 SERVING	SERVINGS PER DAY	SERVINGS PER WEEK
MUSTARD, CATSUP (1 TB)		
JELLY, JAM, HONEY (1 TB)		
SYRUP (3 TB)		
GELATIN DESSERT (1/2 CUP)		
HARD CANDY (6 PIECES)		
POPCORN, PLAIN AIRPOPPED (1 1/2 CUPS)		
WINE, LIQUOR (1 DRINK)		
BEER, SOFT DRINKS (12 OZ CAN)		
REDUCED CALORIE SALAD DRESSINGS (2 TB)		
CREAM OR CREAMER (1 TB)		
REDUCED CALORIE MAYO; MARG. (1 TB)		
SHERBET (1/2 CUP)		
PRETZELS (10)		
COOKIES (2)		
OLIVES (10)		
SOUR OR WHIPPED CREAM (3 TB)		
CREAM SAUCES, GRAVY (1/4 CUP)		
MARGARINE, BUTTER, MAYONNAISE (1 TB)		
BACON (3 SLICES)		
CREAM CHEESE (1 OZ)		
CHOCOLATE BAR (1 OZ)		
CAKE, PIE (1 PIECE)		
DOUGHNUT (1)		
POTATO, CORN CHIPS		
ONION RINGS (1 ORDER)		
SALAD DRESSING (2 TB)		

APPENDIX C

Body weight and body mass index values for the control and treatment group by gender

Parameter	Control		Treatment	
	male (n=12)	female (n=5)	male (n=8)	female (n=7)
Weight(kg)				
Baseline	77.06 ± 10.69[a]	71.58 ± 3.31	86.94 ± 10.01	66.11 ± 6.22
Post treatment	77.36 ± 10.49	71.58 ± 3.22	84.76 ± 9.89[b]***	64.74 ± 6.19***
Body mass index[c]				
Baseline	25.16 ± 2.82	25.10 ± 1.56	27.10 ± 1.56	23.96 ± 3.05
Post treatment	25.28 ± 2.76	25.08 ± 0.89	26.48 ± 1.73[b]***	23.50 ± 3.23***

[a]Mean ± standard deviation.

[b]Post treatment values for treatment group by gender.

[c]Body mass index(kg/m^2)

*** P<.001